Neftel (W. B.)

CONTRIBUTION

TO

THE ETIOLOGY OF EPILEPSY.

BY

WILLIAM B. NEFTEL, M.D.,

OF NEW YORK.

EXTRACTED FROM THE TRANSACTIONS OF THE

INTERNATIONAL MEDICAL CONGRESS,

PHILADELPHIA, SEPTEMBER, 1876.

PHILADELPHIA:
1877.

CONTRIBUTION

TO

THE ETIOLOGY OF EPILEPSY.

BY

WILLIAM B. NEFTEL, M.D.,

OF NEW YORK.

EXTRACTED FROM THE TRANSACTIONS OF THE

INTERNATIONAL MEDICAL CONGRESS,

PHILADELPHIA, SEPTEMBER, 1876.

PHILADELPHIA:
1877.

PHILADELPHIA:
COLLINS, PRINTER,
705 Jayne Street.

CONTRIBUTION TO THE ETIOLOGY OF EPILEPSY.

THE remarkable progress made in our knowledge of epilepsy commences with the classical researches of Brown-Séquard in 1850.[1] As the results obtained by him are well known to the profession, I shall merely mention here a few of the more important points. After the division of one half of the spinal cord, or of one sciatic nerve, in the guinea-pig, the animal becomes epileptic in the course of several weeks, exhibiting on the injured side the so-called epileptogenic zone. This zone is characterized by a lowered sensibility, and is situated below the eye, especially at the angle of the lower jaw, and extending toward the neck and scapula. If the section be made through the entire cord, or through both sciatic nerves, the epileptogenic zone appears on both sides, and if the pedunculus cerebri be injured, the epileptogenic zone appears on the opposite side. The epileptic attacks sometimes occur spontaneously, but they can be called forth at any moment by irritating, or even touching, the epileptogenic zone. The artificially produced epilepsy is transmitted by inheritance to the descendants of the animal on which the experiment has been performed.

These facts are of immense importance for the study of epilepsy in general, though as yet they do not admit of a direct application to human pathology, with the exception of the hereditary disposition, which, however, had been already previously established. It is easy to presume that epilepsy can be brought on by injury of a sensitive or of a mixed nerve, and therefore also by injury of the sciatic nerve, or of the spinal cord, but it is not in accordance with the daily experience of physicians that affections of the spine or of the sciatic should cause epilepsy, with an epileptogenic zone. Thus amputation of the thigh (section of the sciatic) is not followed by epilepsy, with an epileptogenic zone. Again, W. Müller[2] has published a case in which the injury of the cord in the human subject much resembled the experiments of Brown-Séquard on guinea-pigs. A man received a wound in the back from a knife that penetrated through the cord; the patient lived forty-three days, exhibiting all the symptoms of injury of the cord on one side, but having no convulsions whatever, though at the autopsy it was found that the entire left side of the cord had been perfectly divided.[3]

[1] Brown-Séquard, Researches on Epilepsy, etc., Boston, 1857; also Journal de la Physiologie des Hommes et des Animaux, 1858–1860; Archives de Physiologie normale et pathologique, 1868–1872.

[2] Wilhelm Müller, Beiträge zur pathologischen Anatomie und Physiologie des menschlichen Rückenmarkes. Leipzig, 1871.

[3] I omit giving here examples of epilepsy caused by injuries of sensitive nerves, and will only mention that it remains as yet undecided whether the epilepsy in these cases is caused by neuritis ascendens, or by purely functional centripetal excitations. Thus in the case described by Virchow, the epilepsy was caused by an injury of the median nerve (neuritis interstitialis prolifera), and in Echeverria's case by a lesion of the ulnar nerve (neuritis).

In 1871, Westphal,[1] in repeating the experiments of Brown-Séquard, whose conclusions he confirmed in all particulars, found that guinea-pigs could be made epileptic by producing a commotion of the brain. By knocking the animal's skull against a solid object, or by inflicting with a small hammer (such as that used in percussion) one or several blows upon its head, the animal is seized with general tonic and clonic convulsions, quite identical in character with the epileptic attacks exhibited by the animals treated by Brown-Séquard's method. If the commotion is very intense, the blow being too violent, the convulsions are generally followed by the death of the animal from paralysis of respiration, the action of the heart still continuing for several minutes. If, however, the blows are moderate, the animals entirely recover from the first attack, but after the lapse of several weeks exhibit the epileptic state and the epileptogenic zone on both sides, exactly like the animals artificially made epileptic by a bilateral section of the cord or of both sciatic nerves. In young animals an attack is called forth by a few slight percussions with the hammer, while in grown, vigorous animals a considerable amount of force is required to produce the same effect.

When the blows are very slight, no real epileptic attacks follow, but, instead of them, what may be called abortive attacks, consisting of peculiar reflex movements. If one of the epileptogenic zones be pinched in such an animal, it scratches the place with the corresponding extremity, and, closing the eyes, turns the head toward the same side. Animals exhibiting abortive attacks can easily be made epileptic by repeating the same operation. The epileptic state and the epileptogenic zones appear generally four or five weeks after the commotion, and may last six months or longer, after which they gradually disappear, though a slight blow on the head can easily bring them on again. Sometimes the attacks manifest themselves spontaneously, but they can always be called forth by mechanical irritation of the epileptogenic zone on each side. The epilepsy artificially produced by cerebral commotion is also transmitted by inheritance.

In order to ascertain the anatomical lesion which caused the epilepsy after these experiments, Westphal removed the skin and periosteum, and, inflicting the blows directly on the bones of the skull, obtained the same result, viz., an immediate attack, followed later by the epileptic state and the epileptogenic zones. This shows conclusively that the sensitive nerves of the skin or of the periosteum have nothing to do with these phenomena. Moreover, the autopsy made by Westphal immediately after the first attack following the blow, did not reveal any lesion either of the skull, or of the cerebral hemispheres, or of the cerebellum. He found only capillary hemorrhages in the gray and white substances of the medulla oblongata and of the upper cervical portion of the cord. Though the experiments of Westphal apparently have some similitude with traumatic injuries of the head, in man, yet he rejects any application of the results of his experimental researches to human pathology.

On the other hand, in Billroth's case there was no neuritis, though the epilepsy was induced by a traumatic cause affecting the sciatic nerve. In the same way wounds and cicatrices may induce epilepsy by the irritation of sensitive nerves. Thus in a case described by Schnee, the epilepsy was brought on by a wound of the head, which left a very sensitive cicatrix the slightest touching of which would call forth an epileptic attack. The epilepsy disappeared after the excision of the cicatrix, in which microscopical examination revealed the presence of neuritis.

[1] Westphal, Ueber künstliche Erzeugung von Epilepsie bei Meerschweinchen; Berl. klin. Wochensch., 1871, No. 38, 39.

He says: "I am not acquainted with a single observation of cerebral commotion (in the human subject), in which convulsions are mentioned among its symptoms."[1]

Hitzig[2] has also produced epileptiform convulsions, in dogs, by injuring the cerebral cortex. There is no doubt that epileptiform convulsions can be called forth in various ways, especially by injuring sensitive parts, among others therefore some central nervous structures. But the epileptiform convulsions thus produced are by no means identical with epilepsy, which, besides convulsions, requires the presence of the so-called epileptic state. In speaking therefore of real epilepsy, we must exclude such convulsions as are caused by the sequelæ of injuries of the cortex (hemorrhages, inflammation, softening, etc.), as in the experiments of Hitzig, or in the cases of Hughlings-Jackson.[3] These latter seem also to have depended on lesions of the cerebral cortex, whereas the genuine epilepsy may exist without any palpable textural changes of the brain.

The following case, lately observed by me, presents unusual interest, inasmuch as it resembles in many respects the experiments on animals, and has besides the great advantage of giving in a precise manner the subjective feelings of the patient. It is not only interesting as regards symptomatology and treatment, but especially so as throwing some light on the etiology of epilepsy.

Mr. H. W. K., lawyer, 24 years old, of a healthy family, without any hereditary neuropathic disposition, was formerly quite well, intelligent, industrious, and very regular in his habits. During a riot in Washington, in July, 1869, a negro struck him with a loaded cane on the head. The first terrible blow brought him senseless to the ground, where he received several additional blows on the head. He was carried to his house in an unconscious state, and, according to the opinion of the attending physicians, remained during three days and three nights in a comatose and hopeless condition. The first blow, which rendered him senseless, was struck on the right side of the forehead, in the direction from the tuber frontale toward the right crista frontalis externa and linea semicircularis. Fortunately the patient had long hair at that time, and wore a straw hat, which undoubtedly weakened somewhat the force of the blows. To the same circumstance must be ascribed the absence of any injury to the skull, on which no marks of contusion were noticeable. The greatest danger that threatened the patient's life was the exceedingly feeble, scarcely perceptible respiration, which at times seemed entirely extinguished. The action of the heart was also very weak (pulse 20 in a minute), and immediate death was undoubtedly prevented only by the strenuous efforts of Surgeon-General Barnes, who remained day and night at the patient's bedside, energetically using all possible means of excitation and derivation.

On the fourth day the consciousness gradually returned, though the patient still remained motionless, and therefore was still considered unconscious. He could, however, hear and understand everything, and felt the greatest fear of being buried alive, hearing the physicians express their opinion of his hopeless condition, and of the fatal termination which must necessarily soon take place. At the end of the fourth day, he felt in the extremities a pricking sensation (formication), which gradually extended all over the body, and slowly disappeared on the fifth day. At the same time he began to make feeble movements with the extremities, and soon was able to contract all other voluntary muscles; but he could not swallow or speak until the end of the first week. During the second week after the commotion, the patient experienced the

[1] Loc. cit. p. 462.
[2] Hitzig, Untersuchungen uber das Gehirn. Berlin, 1874, S. 270.
[3] Hughlings-Jackson, West Riding Lunatic Asylum Medical Reports, vol. iii., 1873.

greatest inclination to sleep, and could not awaken spontaneously. According to the advice of his physicians, he was awakened at times, and prevented from sleeping continuously. He was extremely sensitive to noises, easily felt exhausted, and after the slightest exertion would become unconscious, and remain so during ten minutes, exhibiting at the same time convulsive movements. He remained in bed during three months, having the epileptic attacks at first very often, and then once in two or three days. At last his health commenced to improve, he had a better appetite, and could exercise in the open air. But even in the street he was seized several times by an attack, with pallor, loss of consciousness, and epileptic convulsions.

The most distressing symptom the patient suffered from was an intense headache, which commenced with the return of consciousness after the commotion, and has scarcely ever left him during six years. The seat of the pain is the right side of the forehead and the right eye, the spot of the intensest pain corresponding to the right crista frontalis externa, where it passes into the linea semicircularis. The pain at times increases spontaneously, and becomes unbearable, continuing so for days and even weeks, with but short remissions, and finally disappearing almost entirely. At the height of the paroxysm, the right eye becomes absolutely blind (the patient not being able to count fingers), the slightest touch of the painful zone is unbearable, and pressure upon it calls forth loss of consciousness, and convulsions, considered as epileptic by physicians who have witnessed them. Occasionally such fits occur spontaneously, without any pressure upon the painful zone, and the patient sometimes feels the approach of the attack, and has time to lie down carefully, though more frequently it comes on suddenly, and the patient falls without having taken any precaution. All the time the patient suffers from the violent headache, there is a complete analgesia of the painful zone, and the whole forehead appears pale and cold.

When the pain is severest, the patient lies motionless, suffering from nausea and vomiting, and scarcely able to eat anything, as these symptoms are invariably increased by food. The patient passes sleepless nights through the whole duration of the paroxysm, only getting occasional short naps, from which he awakens with increased pain. At last, under the constant use of hot applications to the head, the pain gradually disappears and the patient slowly recovers. He then remains in a comparatively healthy condition for days, weeks, or even months, with good appetite and the power of sleeping, and with the vision of both eyes normal. Nevertheless the painful zone remains sensitive, even during the intervals between the paroxysms, and the slightest touch, and especially pressure, can bring back the paroxysm of pain with all its distressing symptoms, the fainting, and the epileptic convulsions. The patient has always to avoid touching the painful zone, even while washing his face or wearing his hat. He can hardly read, his head and eyes often feeling heavy; he has also to avoid the rays of the sun, and every physical and mental exertion, and as a consequence, has been obliged to give up every occupation. Of late the paroxysms of pain have become more frequent; his memory, that used to be excellent, has begun to fail; he cannot always control his ideas with the former precision, and at times they appear so confused that he fears the development of insanity. In the course of the last three years, though he has been treated by many able physicians, and has used among other remedies a great deal of bromide of potassium and narcotics, nevertheless there has been a progressive aggravation of the disease, especially as regards the intellectual sphere and the frequency of the paroxysms of spontaneous pain.

Present condition: May 29, 1876. The patient is of middle size, with a well-developed skull and chest, and moderately developed muscles and panniculus adiposus; the color of the skin and of the visible mucous membranes is rather pale; the thoracic and abdominal organs are normal; pulse 70; respiration 18; temperature 37° C. He is now suffering from a comparatively mild attack of spontaneous pain, and has scarcely slept or taken any food during several days.

The painful zone extends over the right half of the frontal bone, the anterior lower portion of the right parietal bone, the upper portions of the greater wing of the sphenoid bone, and of the squamous part of the temporal bone, the right eyelids and eye. The slightest touch of this region cannot be borne, and the patient involuntarily shrinks from the approach of the exploring finger. The centre of the intensest pain (spontaneous, as well as produced by pressure) is the upper portion of the crista frontalis externa, and the adjoining lower portion of the linea semicircularis, extending about four centimetres in length, and half a centimetre in width. The right tuber frontale is also more tender than the rest of the painful zone, but there is no particular tenderness over the foramen supraorbitale. The painful zone does not extend below the eye. The patient is unwilling, for the sake of an experiment, to have the slighest pressure made upon the painful zone, but assures me that it has been done many times by mistake or carelessness, and has always been immediately followed by the intensest pain, with pallor of the face, loss of consciousness, and epileptic convulsions. The skin of the forehead is cooler than the rest of the face, it is quite pale, and analgesic; a prick with a needle is not felt. The right eye looks smaller than the left, and is almost blind. (Only quantitative light can be ascertained.) The right pupil is somewhat larger than the left, and reacts sluggishly; the right retinal vessels are narrower than those of the left side. The patient speaks very little, and almost without contracting the facial muscles, or moving the eyes; it seems as if he preferred to turn the whole head, instead of contracting the mimical and eye muscles. His physiognomy therefore lacks expression, or rather expresses stupor, though it is evident that he keeps the facial muscles immovable in order not to increase the pain. The patient has passed a perfectly sleepless night, and complains of nausea, general prostration, and heaviness of the head and eyes.

As every kind of medication that had been resorted to had utterly failed to benefit the patient, I decided to make a careful trial with the galvanic current. I applied the cathode of a weak current (two elements of Siemens) to the nape of the neck, and wanted to apply the anode to the painful zone, but the patient immediately turned away his head, before the electrode had touched the skin. Leaving the cathode at the same place, I then applied the button-shaped anode to the right auriculo-maxillary fossa, and, without interrupting the current, gradually increased its intensity. When six Siemens's elements had been introduced into the circuit, the patient remarked that the pain in the forehead began to increase, whereupon I slowly diminished the intensity of the current, the whole sitting lasting a little over a minute. He felt somewhat better after the treatment, and slept that night until 3 A. M., when he awoke with headache and nausea, the right eye being perfectly blind.

May 30. I tried again the galvanic current, but this time avoiding, as much as possible, fluctuations of the current-intensity by means of a rheostat, intercalated as an accessory current. The current was derived from ten Siemens's elements, the flat cathode being applied to the nape of the neck, and the button-shaped anode to the right auriculo-maxillary fossa; the resistances were gradually augmented in the rheostat, until the full current-intensity had passed through the body during fifteen seconds, after which the current was again slowly diminished by means of the rheostat, and imperceptibly broken. The effect of this treatment was quite surprising. As soon as the current began to flow with full intensity, the patient exclaimed that the pain had entirely left him, the head felt quite free, and the eyes became clear. The sitting had lasted about two minutes. The patient was quite overcome by the sudden disappearance of all the morbid symptoms; his features became movable, and the blindness vanished. He felt quite well the whole day, had a good appetite, could walk a great deal, and slept six hours. Though he awakened without pain, still, the next day, the head and eyes were heavy, and he felt the premonitory symptoms of an approaching paroxysm.

May 31. I repeated the same treatment, and with the same result; the

patient feeling invigorated, as if, according to his own expression, awakening from a long and refreshing sleep. I found on examination (which I had omitted the previous day), after the treatment, the following facts: Slight touching of the painful zone, with the exception of the most sensitive part (crista frontalis, etc.), could be easily borne; there was no trace of anæsthesia or analgesia in this region, but, on the contrary, a prick with a needle was most painfully felt (hyperæsthesia). It was quite surprising to find complete anæsthesia and analgesia of the left (healthy) side of the forehead, extending exactly to the median line. Moreover, on this side differences of temperature could not be distinguished by the patient, and on applying a piece of lint dipped in hot water, and another in iced water, he felt no difference on the left, healthy side, and could easily bear them both; whereas, on the painful zone, he could instantly and with great precision discover the least difference of temperature, the extremes of which were felt most unpleasantly. I ascertained, besides, that the left frontal muscle was paralyzed, the skin of the left (healthy) side remaining smooth when he wrinkled the forehead; also, that the left upper lid could not be perfectly closed.

June 1. I re-examined the patient, and convinced myself again of the correctness of the above-mentioned facts. I repeated daily the above-described treatment, applying in addition the button-shaped anode also to the left fossa auriculo-maxillaris.

June 20. The patient slept ten and a half hours, last night, and the foregoing nights, six or seven hours, which for years he has been unable to do. He feels quite well and strong, and has no pain; the head is clear, the vision of both eyes normal, and he can read very well, has a good appetite, and is able to walk considerable distances. On examination I find both eyes equal in every respect, and the vision normal. The painful zone admits of a moderate pressure, and even the most sensitive spot can be touched, though only very gently. The sensibility and sense of temperature in the painful zone still remain exalted (hyperæsthesia), and there is no trace of analgesia. However, a stronger pressure cannot be borne, especially by the most tender portion of the zone, and the patient still wears his straw hat obliquely toward the left side of the forehead, thus leaving the right side uncovered. The left side of the forehead cannot be corrugated, and is still analgesic, though a slight touch with the finger can be felt; differences of temperature cannot yet be distinguished.

The galvanic treatment was continued daily as before, viz.: flat cathode at the nape of the neck, and button-shaped anode at first to the right, and then to the left, fossa auriculo-maxillaris. Current-intensity seven to ten Siemens's elements, increased and decreased cautiously, though fluctuations of one element could now be easily borne. Current-duration about one minute, or one minute and a half, for each side.

July 3. The painful zone can now bear a considerable pressure, and even the most sensitive portion can be touched. The left side of the forehead can be wrinkled, though less than the right side, and the left eyelid can be perfectly closed. The senses of touch and temperature on this side have considerably improved, but are not yet restored. The analgesic area has been progressively contracting; the patient at first could feel the prick of a needle only at the lower part of the median line, then at the arcus superciliaris, and now the region of the left temple alone remains analgesic. The patient complains of no pain; all his functions are normally discharged; and his general health is quite satisfactory.[1]

I shall not enter into an analysis of the above described symptoms, as I had not the opportunity of observing the patient at the time of the accident, nor immediately after. Although the intelligent patient described with great minuteness all his subjective feelings, and as much as

[1] After thirty-one sittings of galvanic treatment the patient suddenly and unexpectedly discontinued his attendance.

possible gave the opinions of the physicians who then attended him; still we entirely miss the result of an objective examination as regards sensibility and motility; the ophthalmoscopic appearances; the condition of the pupils; the presence of sugar, albumen, or blood in the urine, etc. The patient did not even know during six years that the left (healthy) side of the forehead was anæsthetic and analgesic, with loss of the sense of temperature and with impossibility of wrinkling it (paralysis of the frontal muscle). On the contrary, he considered the painful zone as being analgesic, which may have been the case during the paroxysms of spontaneous pain, though during the intervals I always found this zone hyperæsthetic.

There can be no doubt, however, that the injury produced a cerebral commotion of the gravest kind, which threatened to terminate with paralysis of respiration, exactly as in the experiments of Westphal, when the blow on the head was too violent. This termination was prevented in our case merely by the energetic and successful interference of the attending physician. Whether with the cerebral commotion some transitory affection of the brain had taken place, cannot be decided with certainty. Thus the vaso-motor phenomena might have been ascribed to a lesion on the surface of the cerebral cortex, in which Hitzig[1] and Eulenburg and Landois[2] have quite recently discovered the existence of vaso-motor apparatus. Again, the existence of pachymeningitis might have been assumed with still greater probability in consideration of the somnolence of the patient during the second week after the accident, and the diagnostic value which Griesinger[3] attaches to this symptom, especially when it is found in conjunction with stupor, contracted pupils, and headache. On the other hand, the sudden and almost complete disappearance of all the morbid symptoms after galvanization, and their periodical absence, make very plausible the assumption that no permanent or grave lesion of the brain had been caused by the accident. This supposition is supported by the researches of Fischer,[4] who has shown that a mere traumatic reflex paralysis of the cerebral bloodvessels is sufficient to produce the phenomena of commotion of the brain. From the history of the disease, we have every reason to presume in our case the existence of such a reflex paralysis of the cerebral bloodvessels and not a permanent structural lesion of the brain.

This case therefore elucidates a most important fact, namely, that *epilepsy can be produced in a perfectly healthy person* (having no hereditary neuropathic disposition), *by an injury of the head causing commotion without any structural lesion of the brain.*

Although the analogy of our case with the experiments of Westphal is very striking in many particulars, yet there are also apparently some differences. Thus we find a zone in our patient, the mechanical irritation of which calls forth at any moment loss of consciousness with epileptic convulsions, and though this zone is here hyperæsthetic, while it is anæsthetic in the animal, yet during the paroxysms of spontaneous pain it also appears anæsthetic and analgesic. We must bear in mind that we generally judge of the presence of anæsthesia in animals by the absence of expression of pain (screaming); but this was also the case

[1] Hitzig, Centralbl. für d. med. Wiss., 1876; No. 18.

[2] Eulenburg und Landois, Ueber thermische von den Grosshirnhemisphären ausgehende Einflüsse; Centralbl., 1876; No. 15.

[3] Griesinger, Archiv der Heilkunde, 1862.

[4] H. Fischer, Ueber die Commotio Cerebri; Samml. klin. Vorträge, Leipzig, 1871.

with the patient, who, though suffering intensely when the painful zone was touched, never manifested any sign of pain, but only became pale and lost consciousness. Possibly animals artificially made epileptic, suffer also from paroxysms of pain similar to those of our patient, but which have escaped our observation. Again, the blindness during the paroxysms of pain (probably a vaso-motor phenomenon) has not been noticed in the experiments on animals. Furthermore, the painful zone in our patient was above the eye, whereas in the animal the epileptogenic zone is situated below the eye. These differences, however, do not seem to be of great importance, and may depend on the incompleteness of the observations, on some unknown anatomical variations, or on differences in the localities to which the traumatic irritations are applied.

Of late, cases of epilepsy have been published, in which an epileptogenic zone has been observed, though in by far the great majority of cases this zone is not present. I treated a patient affected with epilepsy, in 1868, who in several respects resembled the one described in this paper, especially as regards the blindness and headache.[1] I regret not to have paid at that time the necessary attention to the existence of an epileptogenic zone. Subsequently I received additional information from this patient, who is now in perfect health, that in his case there was also great tenderness in the region of the temples, which could not be touched, and that the affection originated from an injury of the head. I may add that, in this case too, the disease, which did not yield to the different remedies employed, nor even to a fortnight's galvanization of the head, was successfully treated by a method similar to the one above described. It seems likely that the cases of epilepsy with an epileptogenic zone are of traumatic origin, and it is therefore advisable in every case of epilepsy to look carefully for an epileptogenic zone. Of course, in the human subject the place of this zone may not always correspond to that observed in animals. It is also probable that in the course of time the epileptogenic zone becomes reduced, and finally disappears, thus escaping further observation.

We are now led to inquire whether, in the majority of youthful epileptics, the disease has not been caused by frequent injuries (falling upon the head), in childhood, especially in individuals with a hereditary neuropathic disposition. From this point of view it would seem surprising that comparatively so few are affected with epilepsy and petit mal. The experiments on animals teach us, however, that not every blow upon the head even in the guinea-pig—this neuropathic animal *par excellence*—is liable to produce epilepsy. For the same reason, in the child, the favorable conditions for producing epilepsy are comparatively rare: it would possibly be requisite to have the head fixed to a solid immovable object, striking it repeatedly with a certain force, and perhaps even at a certain region. If we admit the traumatic origin of epilepsy, we are then enabled to explain satisfactorily many phenomena which accompany this disease, as for instance the prevalence of epilepsy in youth, the hereditary transmission of the disease, the negative results obtained from the pathologico-anatomical investigation of the brain (at least the absence of structural lesions of the brain), the vaso-motor and other phenomena which epilepsy has in common with cerebral commotion, etc. Lastly, I should like to direct the attention of the profession to the above-

[1] Neftel, Galvano-Therapeutics. New York, Appleton, 1871, p. 113.

described method of treatment, which may possibly be also found useful in other cases of epilepsy.

To conclude, I shall sum up what has gone before in two propositions:—

I. It is made evident by the case described, that in a perfectly healthy person, free from any hereditary disposition, epilepsy can be brought on by traumatic influences upon the head, causing cerebral commotion without any structural lesion of the brain.

II. Further investigations will be needed to prove that such traumatic influences during childhood may constitute a most frequent etiological factor in the production of epilepsy.